LOW TENSION GLAUCOMA

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The enigma of low tension glaucoma has existed since von Graefe (1857) described "amaurosis with excavation" which had all the ophthalmoscopic features of chronic simple glaucoma without elevation of the intraocular pressure. It must be noted that intraocular pressure in his time was checked by digital tonometry. Von Graefe later withdrew the concept that all excavation of the optic nerve head was due to elevated intraocular pressure.

The introduction of indentation tonometry by Schiotz (1905) established the concept that optic nerve head excavation and atrophy could be present without elevation in the intraocular pressure.

The pathology of both glaucoma and low tension glaucoma was found by Schnabel to be a cavernous or lacunar atrophy of the optic nerve. It is now known that lacunar changes in the optic nerve and brain are due to disturbances of the circulation in small blood vessels. Diurnal pressure variations have been demonstrated to be responsible for some cases of low tension glaucoma.

Congenital malformation of the optic nerve head (Fuchs) and pressure on the optic nerve from tumours of the pituitary and adjacent structures were shown to lead to excavation and atrophy of the optic nerve head. It was believed that calcification of the intracranial portion of the carotid artery pressing on the optic nerve was responsible for some cases but this change has been demonstrated in up to 50% of all the people with no corresponding changes in the optic nerve head. The association of calcified carotids with small vessels feeding the optic nerve was suggested.

Eyes with a low coefficient of scleral rigidty (usually myopic eyes) were found to have higher intraocular pressure by applanation than by indentation tonometry (Winstanley 1959) which accounted again for some patients with this anomaly (L.T.G.).

The introduction of tonography in 1950 (Grant) saw the division of low tension glaucoma into those patients with normal outflow facility and those with chronic simple glaucoma in whom the introcular pressure was lowered by a diminution in aqueous production arising from the compensatory or atrophic changes in the ciliary body.

Low tension glaucoma was found in 0.15% of a total population study (Hollows & Graham). Smal arcuate scotomata were demonstrated in patients whose highest pressures were always below 20mm of mercury (Armaly 1966). Factors other than the intraocular prressure must be incriminated in the production of visual field changes in the arcuate area. After the steroid provocative test was introduced, it appeared that 40% of patients with low tension glaucoma responded, with a high rise in the intraocular pressure (Armaly 1967).

Reduced resistance of the optic nerve to intraocular pressure due to systemic factors was suggested (Haas 1962). The presence of a low perfusion pressure or a history of transient but severe hypotensive episodes in patients with low tension glaucoma is significant. Harrington (1959) sug-

gested that low tension glaucoma was due to a decreased blood flow, which was produced by arteriolar sclerosis and partial occlusion of the optic nerve vessels.

The animal experiments of Hayreh verified that imbalance between the intraocular pressure and systemic blood pressure altered the perfusion of the ciliary circulation and the circulation in the optic nerve head. He studied the effects of lowering the blood pressure at different levels of intraocular pressure:

Systolic Ophthalmic Artery Pressure = 0.80 x systolic B.P. -8.63 ± 3.8

Diastolic Ophthalmic Artery Pressure = 0.80 x diastolic B.P. $+6.95 \pm 3.4$

When the difference between the diastolic blood pressure and the intraocular pressure is 10mm of mercury or less, there is very poor filling of the vessels in the optic disc, seen only in the temporal sector on I.V.F.

When diastolic blood pressure is lower than intraocular pressure, there is no filling of the vessels in the optic disc or choroid. (The capillary circulation at the optic disc appears to be more precarious with a relatively high systolic B.P.) This indicates that the extent of filling of the vessels in the optic disc, peripapillary choroid and the remaining choroid depends upon the difference between perfusion pressure in the ciliary circulation and the intraocular pressure — the greater the difference, the better the filling and vice-versa. From this study low tension glaucoma represents a group showing a fall in perfusion pressure without a rise in the intraocular pressure, resulting in vascular insufficiency similar to that caused by a rise in the intraocular pressure with normal perfusion pressure (glaucoma). Hayreh concludes that glaucoma and low tension glaucoma are identical processes producing ischaemia of the optic disc and peripapillary chorid. The low perfusion may result from narrowing of the posterior ciliary, ophthalmic, Internal Carotid artery or from systemic arterial hypotension.

In Drance's series, 93% of patients with low tension glaucoma showed systemic abnormalities and haemodynamic crises; and and low blood pressure occurred statistically significantly more frequently in them. Such crisis produce the clinical picture only in the elderly and practically never in the young. One must wonder therefore about an association of small or large vessel changes which occur in the elderly and practically never in the young. As damage occurs in only one eye after a crisis, there must be reasons for this asymmetry. These may be a slightly higher intraocular pressure on the damaged side of a partial stenosis of the carotid artery, or may be even local changes in the small vessels feeding one optic nerve head which cannot be recognised clinically.

Low systemic blood pressure is also significantly more common. The blood supply in the optic nerve is favourably influenced by an adequate perfusion pressure (ophthalmic artery pressure less intraocular pressure) and adversely by local vascular disease. The blood supply of the nerve in a person with a low blood pressure, particularly if there is in addition a partial stenosis of the carotid, ophthalmic, or one of the posterior ciliary arteries, may be susceptible to small changes in the intraocular pressure which — while statistically normal for a population — may be twice as high as the intraocular pressure which prevailed in the same individual when he was younger.

The finding of small haemorrhages on the optic disc are usually transient and easily missed. Such haemorrhages always accompany ischaemic optic neuropathy (Foulds 1969) but in these latter patients infarction is usually total or subtotal, and the patients present themselves because of central visual loss or, more often, an altitudinal field loss. The patients with glaucoma or low tension glaucoma have no complaints. In both conditions the haemorrhages probably indicate small episodes of infarction or vascular insufficiency to optic nerve tissue. After such haemorrhages, notching of the involved neuroretinal rim has been described (Begg & Drance 1971) and occurs some two to three months after the haemorrhage has disappeared. The optic nerve after the usual ischaemic optic neuropathy becomes atrophic but rarely cupped.

There are probably many mechanisms by which an optic nerve head may become cupped and atrophic. Some ways have been observed on groups of patients::

(a) One had a pre-existing large cup which was followed by atrophy of a segment of the neuroretinal rim after a local vascular occlusion;

(b) Notching of the neuroretinal rim after the apearance of a small haemorrhage on the disc with the appearance of a corresponding nerve fibre bundle defect, and considered to be a small optic nerve infarction .

(c) A third mechanism may be inferred; a man with deep cupping of both discs developed optic atrophhy on one disc after a massive gastro-intestinal bleed.

The presence of cupping in an atrophic disc does not necessarily differentiate an acute from a chronic vascular impairment.

Low tension glaucoma does not always develop progressive field defects. The lack of progression in almost all cases who had a Lumo-dynamic crisis — 99% in Drance's series — and the progression in those who did not have such a crisis (70%) is an important finding and can be valuable in estimating prognosis. The presence of low blood pressure, vascular disease, high myopia and diabetes make slow progression of field defects likely.

Mechanical Factors:
(Phillips, Tomlinson, Leighton.)

The high cup/disc ratio with which a high axial length of the eyeball is associated as well as ocular tension may explain the unduly high prevalence of myopic eyes in both open angle glaucoma and low tension glaucoma.

Eyes with low tension glaucoma are significantly more myopic and have a greater axial length than open angle glaucoma. For pathological cupping and glaucomatous field defects to occur in large eyeballs, the ocular tension needs to be only slightly raised. In small eyeballs the critical level of ocular tension for the development of field defects needs to be higher to offset the lower susceptibility of the optic disc.

The difference in axial length, which would tend to make low tension glaucoma eyes relatively more myopic than open angle glaucoma is neutralised by a significantly greater vertical corneal radius in eyes with low tension glaucoma.

A signficantly greater length of the vitreous body in low tension glaucoma more than open angle glaucoma indicates that the posterior segment of the eye is bigger and would tend to have a higher cup/disc ratio and therefore more susceptible to glaucomatous cupping, with the result that the ocular tension needs to be only slightly raised to produce cupping. Sjogren (1946) had observed that deeper excavation of the optic disc is found in low tension glaucoma relative to open angle glaucoma.

The retinal nerve fibres in an eye with a greater axial length and a high cup/disc ratio and probably a large disc may have less 'slack', especially at the disc, than in a normal eye; so that quite a small degree of cupping may stretch them or kink them over the edge of the disc or the cribriform plate. That there seems to be more 'slack' at the upper half of the disc may explain why the lower field is normally spared until late in the disease; the papillo-macular bundle may suffer less damage because it passes through the cribriform plate quite peripherally. Another related factcor may be the tendency for the blood supply of the inferior half of the disc to be less well developed than the upper half because the foetal fissure is located in an inferior position.

The large disc which exists in the eye with a greater length may be a factor in making it more suscecptible to pathological cupping than the normal or small disc. The 'force' tending to bow the disc backwards depends primarily on the pressure x area (a 10% increase in diameter will produce a 21% increase in area.) The bowing is the result of (a) bending and (b) stretching, the effect of the area being greater on the former.

Treatment

The treatment of low tension glaucoma is directed towards reduction in the ocular

tension by medical or surgical methods. One must bear in mind that it may be more beneficial to reduce the intraocular pressure from 40 to 25 than from 20 to 15. As it is more difficult to obtain a large reduction in the intraocular pressure if the initial pressure is low, the possibility of improving disc perfusion is much less.

In low tension glaucoma we have an ischaemic process of the optic nerve head which may be the result of many interacting factors. The search for a single cause or process which leads to this condition may therefore continue to be frustrating. Even the separation of low tension galucoma from glaucoma proper is artificial and to some lacks significance. It seems unreasonable to think of two separate diseases occurring in those patients with classical low tension glaucoma in one eye and overt glaucoma in the other. Chronic simple glaucoma and low tension glaucoma are more likely to be manifestations of a disease process in which many factors assume varying importance in interfering with the perfusion of the optic nerve head. A clear recognition and understanding of the factors concerned in producing low tension glaucoma, and the signifiance of the intraocular pressure is an important, but by no means the only, factor leading to the ischaemia of the optic nerve head which may help to answer some of the uncertainties of chronic simple glaucoma itself.

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